AN ANALYSIS OF OPHTHALMIC MANIFESTATIONS IN AIDS PATIENTS IN LIEU OF THEIR CD4+T LYMPHOCYTE COUNT.

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CERTIFICATE

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INTRODUCTION

Human fight against emerging dreaded diseases like Tuberculosis, Syphilis and Leprosy and Malignancies is continuing for decades. Now we face still more challenging, disastrous and dangerous disease, AIDS, with its capability of destroying one's own immune mechanism. Destroying all boundaries among the nations, races and religions, this disease spreads in a very high speed.

Emerging as a global pandemic, this killer disease infected tens of millions of patients in less than three decades and orphaned more than ten million children in the same period. The HIV epidemic continues to increase at an alarming rate. The countries with large population and areas of poverty contribute the bulk of the cases.

Acquired immunodeficiency syndrome (AIDS) is an infectious disease caused by a retrovirus, the Human Immunodeficiency Virus (HIV). This syndrome is characterized by a gradual decrease in circulating CD4 + T lymphocytes and subsequent development of various opportunistic infections and neoplasia.

At present, an estimated population of 40 million adults and 3 million children are infected with HIV, and 25 million people had already died of AIDS. There are 13000 new infections everyday and current trends suggest that there may be 90-110 million people will be affected by the end of this decade.

World estimates of the HIV & AIDS epidemics at the end of 2005

Number of people living with HIV/AIDS in 2005		**Estimate***	**Range***
	Total	**40.3**	**36.7-45.3**
	Adults	**38.0**	**34.5-42.6**
	Women	**17.5**	**16.2-19.3**
	Children	**2.3**	**2.1-2.8**
People newly infected with HIV in 2005		**Estimate***	**Range***
	Total	**4.9**	**4.3-6.6**
	Adults	**4.2**	**3.6-5.8**
	Children	**0.70**	**0.63-0.82**
AIDS deaths in 2005		**Estimate***	**Range***
	Total	**3.1**	**2.8-3.6**
	Adults	**2.6**	**2.3-2.9**
	Children	**0.57**	**0.51-0.67**

* *millions*

Because of the 100% mortality rate, the worldwide distribution and the enormous social stigma attached to the disease, AIDS, unlike any other past epidemic, requires mobilization of national resources, the support of community leaders and public education regarding safe sex practices, training of health care personnel and help from industry in developing vaccines and newer antiviral drugs.

The role of the ophthalmologist in the diagnosis of AIDS is becoming increasingly important. Not only does the eye reflects systemic disease, but ocular involvement may often precede systemic manifestations. In the AIDS patient, the ophthalmologist thus has an opportunity to make not only a sight saving, but also a life saving diagnosis of disseminated opportunistic infections.

EPIDEMIOLOGY AND HISTORY

AIDS was first recognized in the united states in the summer of 1981, when the Centre for Disease Control and prevention (CDC) reported the unexplained occurrence of pneumocystis carinii pneumonia in five previously healthy homosexual men in Los Angels and of Kaposi's Sarcoma in 26 previously healthy homosexual men in New York and Los Angeles.

In 1983, Human Immunodeficiency Virus was isolated from a patient with lymphadenopathy, and by 1984 it was demonstrated clearly to be the causative agent of AIDS. By 1994, a million cases had been reported to the World Health Organization (WHO) and the pandemic has now affected all continents.

At the end of 2006, according to Joint United Nations Programme on HIV/AIDS, 45.3 million people are estimated to be living with HIV/AIDS (41.5 million adults and 3.8 million children below 15yrs). Out of which 90% of cases are in the economically productive age group of 15-45 yrs.

Since the beginning of the epidemic, there have been as estimated 25 million deaths due to AIDS. Women are becoming increasingly affected by HIV. Approximately 40% or 15 million of the 37.2 million adults living with HIV/AIDS world wide are women.

Once in every three children orphaned by HIV/AIDS is under age five. Since the beginning of the global pandemic, over 13 million under the age of 15 have lost their parents to HIV/AIDS.

DEFINITION AND CLASSIFICATION OF AIDS

Expanded WHO Case Definition for AIDS.

An adult or adolescent (>12yrs of age) is considered to have AIDS if a test for HIV antibody gives a positive result and one or more of the following conditions are present.

- ≥10% of body weight loss or cachexia with chronic diarrhea or chronic fever or both intermittent or continuous for at least one month.
- Cryptococcal meningitis
- Pulmonary or extrapulmonary tuberculosis
- Kaposi's sarcoma
- Candidiasis of oesophagus.
- Clinically diagnosed life threatening or recurrent episodes of pneumonia with or without etiological confirmation.
- Invasive cervical cancer.
- Pneumocystis carinii pneumonia
- Disseminated M.avium infection

- CMV disease
- HIV associated dementia
- Toxoplasmosis
- Immunoblastic lymphoma
- Chronic cryptosporidiosis
- Disseminated histoplasmosis

WHO clinical staging system for HIV infection.

Stage I:

Only a small number of people have a recognizable acute illness at the time of infection with the virus and most are unaware. Initially HIV infection is clinically silent or asymptomatic. Very few patients may develop mild viral fever like illness.

Stage II:

As immunosuppression develops, the CD4 count falls and viral load increases, there is increasing susceptibility to a small range of clinical illnesses. Clinical manifestation may consist of mild weight loss, skin and oral problems recurrent sinusitis or herpes zoster.

Stage III:

With further immunosuppression, the susceptibility widens to select group of common and more virulent infection such as TB and bacterial pneumonia. Common symptoms in this stage are weight loss more than 10%, chronic diarrhea and prolonged unexplained fever.

Stage IV:

With more profound immunosuppression, when the CD4+ T lymphocyte count falls below 200, the opportunistic infections develop and the individuals considered to have advanced disease.

Disease Progression and Survival:

Disease progression and survival with HIV is variable such as Rapid Progressors, Slow Progressors and Non Progressors. It may be rapidly progressive over 2yrs or hardly progresses at all over 15 yrs. However older age and low socio-economic status adversely affect survival.

VIROLOGY – HUMAN IMMUNODEFICIENCY VIRUS

HIV belongs to lentivirus subgroup of the family **retroviridae**. First identified as Lymphadenopathy Associated Virus (LAV), renamed as Human T cell Lymphotrophic Virus III (HTLV – III). In 1986 **International committee on virus nomenclature** decided this new generic name Human Immunodeficiency Virus (HIV).

There are two types of HIV, type 1 and 2. Type 1 is more virulent pathogen than type 2 and seen worldwide. HIV 1 and 2 are each approximately 100nm in diameter and have a **single stranded RNA genome**. The virion has a cylindrical nucleo capsid that contains the single stranded RNA and viral enzymes including **proteinase, integrase** and **reverse transcriptase**. Enzyme reverse transcriptase is the characteristic feature of retrovirus. Surrounding the capsid is a lipid envelope that is derived from the infected host cell and that contains three structural genes; **gag, pol** and **env.** HIV 1 and HIV 2 are genetically similar in the gag and pol regions. The env regions are however different. This variation results in differences in the envelope glycoproteins of these viruses. Such heterogenicity leads to specific immune responses to these viruses, which necessitates different immune assays or western blot procedures for serologic diagnosis of HIV 1 and HIV 2. In addition to the three structural genes, HIV contains six additional regulatory genes, **tat, rev, vif, vpr, ref** and **vpu.** Of these, two (tat and rev) are essential for viral replication. HIV isolates show marked heterogenicity in the env and the ref genes, which results in differing tissue and cell tropism, variation in pathogenesis, disparable responses to therapy and potential challenges to develop a broadly cross reactive protective vaccine.

TRANSMISSION

Four principle modes of transmission of the virus are recognized.

(i) Sexual intercourse – vaginal or anal intercourse in either heterosexual or homosexual.

(ii) Transfusion of infected blood.

(iii) Use of contaminated needles or syringes.

(iv) Vertical – From an infected mother to her baby.

Mode of Transmission of HIV infection:

Type of Exposure	Efficiency in single exposure	% of Total Transmission
Blood transfusion	>90%	3-5%
Perinatal	30%	5-10%
Heterosexual	0.1 – 1.0 %	60-70%
Homosexual	0.1 -1.0%	5-10%
IV Drug abuse	0.5 – 1.0%	5-10%
Health Care	<0.5%	<0.1%

Although the efficacy of sexual mode of transmission is low, it accounts for the commonest mode of transmission because of the absolute number of risk intercourse is heavy.

PATHOGENESIS

Initial events in HIV infection include attachment of the virus to a distinct group of T cells and monocytes / macrophages that display a membrane antigen complex known as CD4. After attachment, the lipid membrane of the virus fuses with the target cell, thereby allowing entry of the viral core into the host cell cytoplasm. This viral core is subsequently uncoated and transcribed by the reverse transcriptase enzyme which results in a complementary strand of DNA. This DNA then becomes double stranded by the action of cellular enzymes, subsequently becomes circular and enters into the cell nucleus, where integration into the host cellular genome takes place by means of a viral endonuclease. The actively infected cell produces many virions by transcription of proviral DNA. The transcription also generates messenger RNA, which in the cytoplasm is translated into HIV specific structural proteins, that are integrated with the core particles. Final maturation of the virus occurs by a process of reverse endocytosis (budding) at the plasma membrane and susbsequent dissemination occurs.

The initial target cells of HIV, namely CD4+T helper cells and macrophages, show different cytopathic effects. The T cells gradually decrease in number from the virus replication leading to immunodeficiency and subsequent secondary opportunistic infections. In contrast, the infected

macrophages, instead of undergoing lysis, harbor the virus and disseminate it throughout the body.

However, their immune-related functions are altered - decreased migration response to chemoaltractants, defective intracellular killing of various micro organisms such as Toxoplasma gondii and Candida, reduced expression of class II molecules, which impairs the processing and presentation of antigen to T helper cells, and excessive production of tumour necrosis factor alpha (by the macrophages) which leads to dementia, wasting syndrome and unexplained fever.

Disease Course

The illness that results from HIV infection varies from one individual to another with several predictable stages that leads invariably to death. In general infected individuals initially experience an acute primary infection, followed by a relatively asymptomatic infection that can include generalized lymphadenopathy. This progresses to symptomatic disease associated with progressive decline in

T-helper cells, and eventually to advanced HIV disease with the development of opportunistic infections or malignancies.

The acute HIV infection lasts for about one to two weeks and is characterized by symptoms typical of a non specific viral illness. Patients then invariably enter into an asymptomatic phase that can last for two or more than

ten years. During this phase the CD4+ T lymphocyte count varies from about 750 to 200 cells / cu.mm (normal count being 600 to 1400 cells / cu mm).

The asymptomatic phase is followed by advanced HIV disease, which may last for up to three years; during this stage the CD4+ T lymphocyte cells decrease to less than 200 cells / cu mm.

DIAGNOSIS

HIV infection is diagnosed by blood tests that detect HIV antibodies.

The tests usually being done are.

- RAPID test
- ELISA test
- WESTERN BLOT test

RAPID and ELISA tests are sensitive, specific and less expensive. These are commonly used for screening purposes in blood banks and at voluntary counselling and testing centres.

WESTERN BLOT test, though more specific, is costly. Hence it is used primarily to confirm the screening test results which turned out positive.

Diagnosis can also be made by testing for HIV antigens through**.**

- Polymerase Chain Reaction (PCR) test
- Viral load assessment tests like NASBA
- P24 Antigen test

The first two tests become positive after 72 hours of infection and the third one becomes positive after 2 weeks of infection.

Indirect Immunofluorescent Assay (IFA), Radio Immuno Precipitation Assay (RIPA), Passive haemagglutination assay and Dot immunobinding assay also are available.

THERAPY

In the management of HIV infection, the prevention of complication involves not only anti retroviral therapy and prophylactic antimicrobials, but also immunization (e.g. HBV, Influenza vaccines and in specific cases other available vaccines) and early disease detection.

Anti-retroviral drugs in current use belong to four classes:

- Nucleoside Reverse Transcriptase Inhibitors (NRTI'S)
- Non Nucleoside Reverse Transcriptase Inhibitors (NNRTI'S)
- Protease Inhibitors (PI's) and
- Fusion / Entry Inhibitors.

NRTI'S and NNRTI'S inhibit reverse transcriptase and this prevents the virus from infecting the cell. PI's inhibit protease, an enzyme which cleaves viral proteins and is important for the final assembly of virions. PI's are the most potent anti-retroviral drugs. Fusion / Entry inhibitors represent the new class of anti-retrovirals. This injectable drugs inhibit HIV fusion with the CD4 cell membrane and is intended for use in patients who have failed with other regimens.

NRTI	NNRTI	PI	FUSION/ENTRY INHIBITORS
Zidovudine		Indinavir	
Didanosine		Ritonavir	
Stavudine	Delvaridine	Nelfinavir	
	Nevirapine		Enfuvirtide
Lamivudine	Efavirenz	Lopinavir	
Zalcitabine		Saquinavir	
Abacavir		Amprenavir	
Tenofovir			

Viral replication occurs at the rate of about **10^{10} virions per day**. No single drug is able to inhibit this degree of viral replication. Therefore when a patient is exposed to one or a combination of two drugs, the continuing viral replication allows drug resistant mutants to develop.

The recommended regimens consist of

- 2 NRTI'S + 1 NNRTI
- 2 NRTI'S + 1 PI
- 3 NRTI'S
- 2 NRTI'S + PI Boosting (2 PI'S)

CD4+ T LYMPHOCYTE COUNT AS A PREDICTOR OF RISK

For years, the CD4+ T Lymphocyte count proved a reliable predictor of the risk of ocular complications of HIV infection. Recently, however the use of Highly Active Anti Retroviral Therapy (HAART) has allowed substantial and sustained albeit incomplete, repopulation of T lymphocytes to occur in many patients. Such observations have raised the question of whether reconstituted T lymphocyte populations are in fact functional, and more specifically, whether the current or the lowest CD4+ T Lymphocyte count is a better predictor of the risk of HIV associated disorders.

Even then the CD4+ T lymphocyte count reflects the immune status of the patients. Low CD4+ T lymphocyte count indicates the poor immune system. This alarms the physician to look for asymptomatic opportunistic infections which are known to occur in these patients. Absolute Lymphocyte Count (ALC), CD8+ T lymphocyte count, and the ratios among all these also reflects the immune status of the HIV patients. Serial evaluation of these counts are

necessary in all these patients to assess the course of disease and to know the effect of treatment.

CD4+ T Lymphocyte count	**Disorders**
< 500 cells / cu.mm	Kaposi's sarcoma Lymphomas
<250 cells / cu.mm	Pneumocystosis Toxoplasmosis Herpes Zoster Ophthalmicus
< 100 cells / cu.mm	Retinal or conjunctival microvasculopathy CMV retinitis Keratoconjunctivitis sicca Cryptococcosis Microsporidiosis HIV encephalopathy Progressive multifocal encephalopathy

OPHTHALMIC MANIFESTATIONS OF HIV DISEASE

Numerous studies reported that around 40-70% of AIDS patients have ophthalmic manifestations. These lesions are divided into four groups.

- Adnexal lesions
- Anterior segment lesions
- Posterior segment lesions
- Neuro - ophthalmic lesions

Adnexal lesions:

- Herpes Zoster Ophthalmicus
- Kaposi's Sarcoma of Lids and Conjunctiva
- Molluscum Contagiosum of Lid
- Squamous Cell Carcinoma
- Burkitt's Lymphoma
- Orbital Cellulitis

Anterior segment lesions

- Dry eyes
- Conjunctival microvasculopathy
- Infectious Keratitis
- Anterior Uveitis
- IRU – Immune Reconstitution Uveitis.

Posterior segment lesions

- HIV Retinopathy
- CMV Retinitis
- Toxoplasma Retinochoroiditis
- Pneumocystis Choroiditis
- Acute Retinal Necrosis
- Progressive Outer Retinal Necrosis
- Endogenous Endophthalmitis
- Bacterial and Mycobacterial Retinitis

Neuro Ophthal Lesions:

- Cranial Nerve Palsies
- Papilloedema
- Papillitis
- Optic Atrophy

Herpes Zoster Ophthalmicus.

HZO presents as painful vesiculobullous dermatitis, results from the reactivation of previously established primary VZV infection. This virus lies dormant in dorsal root ganglion of any sensory nerve and HZO is due to reactivation in the trigerminal nerve ganglion commonly affecting the ophthalmic division of this cranial nerve. This is uncommon in less than 50

years age and is common in young male homosexuals who are immuno - compromised due to HIV infection. The severity of disease is more in HIV patients than normal immunocompetent patients. 5-15% patients will develop sight threatening complications like Conjunctivitis, Keratitis, Uveitis, and Retinitis.

It is treated with IV Acyclovir 10mg/kg body weight thrice daily for seven days followed by Oral Acyclovir 800mg 5 times / day.

Kaposi's Sarcoma:

This is highly vascularised painless mesenchymal tumour affecting the skin and mucous membrane in 25% of HIV Patients. 20% patients have asymptomatic Kaposi's Sarcoma of eyelids or conjunctiva. This may be missed if fornices are not examined properly. It presents as nodules or polypoidal growth. It may be mimicking as chalazion or subconjunctival haemorrhage. Kaposi's sarcoma should be suspected when a HIV patient is having persistent SCH.

Treatment is with radiation, cryotherapy and intralesional chemotherapy with vinblastin, ά–interferon and liposomal daunorubin are also used. In spite of all these modalities of treatment, 100% recurrence is noted in some studies.

Molluscum Contagiosum:

This is more frequent and severe in patients, who are HIV +ve than who are negative for HIV. It is an infection of skin and mucous membrane caused by DNA Pox Virus, involving face and upper body. Usually presents as multiple small painless umbilicated nodules of 2-10mm diameter.

Pressure over the lesion will produce waxy discharge. This may even spread to bulbar conjunctiva.

Treatment is with cryotherapy, curettage, excision therapy or electrocautery. Podophyllotoxin cream also is used as adjunct to cryotherapy.

Squamous Cell Carcinoma:

Human Pox Virus or P53 gene over expression may play a role in the development of this tumour in patients who are infected with HIV. Treatment is Excision, Enucleation or Excentration, depends on the extension of disease at the time of presentation.

Burkitt's Lymphoma:

In patients who are having systemic Non-Hodgkin's Lymphoma either on treatment or in tumour remission develops lymphomatous infiltration of optic nerve and occlusion of central retinal vein. There is dense infiltration of optic nerve and necrotizing vasculitis involving retinal vessels near optic nerve.

Orbital Cellulitis:

HIV patients may develop orbital cellulitis because of their immuno-compromised status. Aspergillus species and Mucor mycosis are the common etiological agents causing cellulitis. Patients present with swollen lids, erythema, pain, congestion of conjunctiva, proptosis, restriction of movements with defective vision. Treatment is by systemic antibiotics, antifungals and supportive measures.

Dry Eyes:

20% of HIV patients develop dry eyes in later stages of illness. This is due to HIV mediated inflammation and damage to major and accessory lacrimal glands. Complaints are red eye, watering, burning and FB sensation. Corneal ulcers may develop as complication. Schirmer's test and Fluoresin dye test are used to assess the severity. Artificial tears containing methyl cellulose are prescribed.

Conjunctival Microvasculopathy:

70-80% of HIV patients have asymptomatic conjunctival microvascular changes. The changes are segmental vascular dilatation, narrowing, micro-aneurysm formation, appearance of comma shaped vascular fragments and sludging of blood columns. Increased plasma viscosity, and immune complex deposition are considered to be the pathological event. Direct infestation of conjunctival vascular endothelium by the virus also is suggested.

Infectious Keratitis:

VZV and HZV are the most common etiological agents. Painful corneal ulceration with characteristic dendritic or branching pattern is noted. Treatment is with oral acyclovir or famciclovir. Long term therapy is needed.

Bacterial and fungal keratitis also are noted. Staphylococcus aureus and Pseudomonas aeruginosa are the common causative agents. Candidal species are common in IV drug users. Microsporidia has also been implicated in some cases with superficial punctuate keratopathy. Gram stain, KOH mount are used to identify the organisms. Microsporidia are seen within the cornea or conjunctival epithelial cells with the use of either Massontrichrome or Giemsa staining.

Iridocyclitis:

Mild Iritis and Iridocyclitis are common in HIV patients and usually associated with CMV retinitis or HZO. Also it is seen with Toxoplasma retinochoroiditis, Syphilitic retinochoroiditis, and in Tuberculous retinitis.

Immune Reconstitution Uveitis:

IRU syndrome can occur in patients with advanced HIV disease soon after initiation of effective HAART therapy, leading to exacerbation of previously treated opportunistic infection or new presentation of a previously sub clinical uveitis, when the CD4+T lymphocyte counts increasing to >100 cells / cu.mm from the previously low levels. Otherwise Cidofovir and Rifabutin also can cause uveitis.

Long term complications of IRU include cataract, vitritis, papillitis, optic disc new vessels or optic atrophy, cystoid macular oedema and vitreous haemorrhage.

HIV Retinopathy:

Like microvasculopathy in conjunctiva, retina also is affected by HIV retinopathy. The pathology is same as in conjunctiva like increased viscosity, immune complex deposition and direct cytopathic effect of virus on retinal vascular endothelium. All these things lead on to interruption of axoplasmic flow which manifests as Cotton Wool Spots. Microaneurysm, Retinal haemorrhages, telengiectasias and capillary nonperfusion also are seen. Exudates are not seen, differentiating this from CMV retinitis. Commonly these findings are seen when the CD4+T lympocyte count falls below 100 cells / cu.mm. These changes are usually asymptomatic and not vision threatening.

But these changes may lead to progressive optic atrophy, with loss of color vision, contrast sensitivity and visual field defects.

Fluorescein angiography shows microaneurysm, blocked fluorescence (in the areas of CWS), telengieclasias and focal areas of non perfusion and capillary loss.

CMV Retinitis:

40-60% of patients with HIV infection show features of CMV retinitis in various studies. This is the most common opportunistic infection and the main cause of blindness in AIDS patients.

Usually the patient's CD4+T lymphocyte count is in between 50-100 cells/ cu.mm, when they are affected by CMV retinitis. It is rare when the CD4+T lymphocyte count is >100 cells / cu.mm. CMV retinopathy may be the first ocular manifestation in more patients and usually patients will develop this within the first 7 years of the disease.

Cytomegalovirus is a DNA virus is Herpes group. There is viral invasion of retinal cells lead to necrosis of retina. The patients complain of reduced vision, floaters, blurred vision, loss of peripheral field or blind spots.

There are two different features seen in CMV retinitis. The common one is described as “ pizza pie” appearance characterized by confluent yellow white areas of necrosis and predominance of hemorrhage. There is full thickness necrotizing retinitis. This lesion commonly found in the posterior pole adjacent to the blood vessels. The second pattern is “ Indolent granular retinitis” also called “brush fire” pattern. There is large central clearance appearing as retinal atrophy with a peripheral white granular border, little or no haemorrhages and no vasculitis. These are commonly seen in retinal periphery. The lesser common one is “frosted branch angitis” type to describe extensive vascular sheathing. There is associated perivasculitis, vascular attenuation and vessel closure.

Progression is usually slow along active border and along blood vessels. These lesions have the tendency to enlarge and coalesce over time. Retina behind the active edge of retinitis became pale and atrophic. Necrotic retina is prone to develop breaks and subsequently develop rhegmatogenous RD if peripheral retina is involved. Secondary neovascular glaucoma may occur in CMV retinitis.

Fluorescein Angiography shows hyperfluoresence starts at centre and spreads to periphery. These areas are smaller than gross lesions.

The main drugs for CMV retinitis are Ganciclovir, Foscarnet and Cidofovir. All these are virostatic, not virocidal. Ganciclovir is a cyclic nucleoside, dihydroxy propoxy methyl guanine.

Ganciclovir is started in induction phase as 10mg/kg bodyweight daily in two divided doses given IV for 2-3 weeks, followed by maintenance dose of 5mg/kg bodyweight daily indefinitely.

Intravitreal Gancyclovir injection also is given as 200mg in 0.1cc once weekly. Intravitreal Ganciclovir implant is commonly used nowadays to avoid systemic complications of the drug. It is inserted via parsplana and sutured to sclera. In this method, vitreal concentration of the drug is two fold more than IV route. Implants deliver the drug slowly for 6-7 months. But it does not give protection against systemic CMV infection or infection in the fellow eye.

Alternatively IV Foscarnet is given 60mg/kg 8hrly for 3 weeks then in daily dosage. IV cidofovir in the dosage of 5mg/kg once weekly for 2 doses then every 2 weeks also is given. Both these drugs can also be given through intravitreal route.

Cidofovir and Ganciclovir causes nephrotoxicity and bone marrow toxicity. Foscarnet is toxic to liver.

Recent studies suggest that anti CMV treatment may be stopped if CD4+ T lymphocyte count is raised more than 100-150 cells / cu.mm. for 3-6 months with suppression of viral load.

Ganciclovir is good in preserving the vision. But if the lesions involve macula or ONH at presentation, the visual progrosis is poor.

Toxoplasma Retinochoroiditis:

This can be seen in CD4+T lymphocyte count <250 cells / cu.mm but usually the CD4+ count is <100 cells / cu.mm. The bradyzoites of Toxoplasma gondii are transformed into tachyzoites that allows infiltration of retina and choroids leads to retinochoroiditis.

Retinal findings are single or multifocal, discrete or diffuse areas of necrosis. This is bilateral in 30-50%. Retinochoroiditis is due to acquired infection rather than reactivation. There is more vitritis. There may be associated intracranial lesions in some patients.

Fluorescein angiography shows hyperfluoresence starts at periphery and spreads to centre and the fluorescent area is larger than the gross lesion.

Both IgG and IgM antibody levels are raised, however in very immunocompromised patients these tests may be negative.

Treatment is with Sulphadiazine, Clindamycin, Pyrimethamine and Folinic acid. Folinic acid is given to counter macrocytic anemia induced by Pyrimethamine.

Acute Retinal Necrosis (ARN)

This is due to VZV, HSV or CMV. In HIV patients with HZO, 17% develop ARN and in 30% it is bilateral. It presents as one or more foci of retinal necrosis with discrete borders and is located in peripheral retina, described as "swiss cheese" pattern. There is prominent inflammation in anterior and posterior chamber and an occlusive vasculitis involves arterioles. Holes may develop later in the necrotizing areas of retina leading to RD. Treatment is with IV Acyclovir or Famciclovir combined with laser treatment to the posterior margins of necrotic areas to prevent RD. Final outcome is hampered by retinitis involving macula and optic atrophy.

Progressive Outer Retinal Necrosis (PORN)

It is a devastating viral retinitis due to VZV. Occurrence of PORN usually relates to recent herpetic infection with low CD4 counts. There is sudden reduction in vision. Retinitis is typically multifocal, posterior and involves deeper layers of retina. Inflammatory signs are absent. Multiple white plaqnes near posterior retinal pole progress rapidly to necrosis leading to retinal detachment. There is no effective treatment and visual prognosis is poor.

In patients with PORN, 66% become blind in 6 weeks of diagnosis despite aggressive treatment. Acyclovir or Ganciclovir may be tried.

Pneumocystis Choroiditis:

Pneumocystis carinii pneumonia is one of the AIDS defining opportunistic infection. It is an unicellular protozoan spreads via blood stream to choroid, commonly from respiratory system. Pneumocystis choroiditis is characterized by multiple yellow white subretinal deposits throughout the posterior pole. Visual symptoms are less as there is no vitritis.

Fluorescein study reveals early hyperfluorescence with late staining. Treatment is with IV Trimethoprim with Sulphamethoxazole or IV Pentamidine.

Fungal Chorioretinitis:

Common agents are Histoplasma and Cryptococcus. Usually there is haematogenous spread from pulmonary system or may be as direct extension from CNS involvement. Fundus shows multiple yellowish white mildly elevated chorioretinal lesions. These may progress to fungal endophthalmitis.

Bacterial and Mycobacterial retinitis:

Syphitis, and M. avium intercellulare infection have been demonstrated in autopsies of some patients with disseminated infection.

Neuro ophthal Manifestation:

Neuro ophthal complications occur in 15-20% patients with HIV infection. Commonly presents as papilloedema, papillitis, cranial nerve palsies, AION and optic atrophy.

50% of neuro ophthal lesions are due to Cryptococcal meningitis. Common presentation is papilloedema bilaterally with associated signs of meningitis. Cranial nerve palsies are reported in 33% cases. III N is commonly affected followed by IV and VI nerves. Common complaint is diplopia.

Causes of raised intracranial tension in HIV are NHL, Toxoplasma encephalitis, Cryptococcal meningitis, and metastasis commonly from Kaposi's sarcoma.

AION is noted in advanced HIV cases.

Neurosyphilis, Toxoplasmosis are reported to cause neuro ophthal manifestations. Neurosyphilis run a more rapid and aggressive course in HIV patients than other immunocompetent patients.

Diffuse encephalopathy may be due to direct effect of virus (HIV encephalopathy) or super infection from polyoma virus causing progressive multifocal leuco encephalopathy.

Optic nerve may be damaged by infarction or direct infiltration by HIV virus.

AIM OF THE STUDY

- To evaluate the various ophthalmic manifestations in proven HIV seropositive patients and document findings
- To find out the correlation between the CD4+ T lymphocyte count of HIV seropositive patients and the nature and severity of the ophthalmic manifestations.
- To study the variations in presentation of ophthalmic manifestations in different levels of immunosuppresion.
- To stress the important role of the ophthalmologist in HIV patients for preventing the visual loss by early diagnosis and instituting appropriate therapy.

MATERIALS AND METHODS

This clinical study was done to evaluate the various ophthalmic manifestations in a south Indian population of HIV infected persons and to find out the correlation between the CD4+ T lymphocyte count and nature and severity of ophthalmic manifestations.

A total of 60 HIV positive patients were studied.

The study period extended from January 2004 to December 2005.

The HIV statuses of the patients were confirmed by ELISA method in laboratories of Government Hospital for Thoracic Medicine Tambaram. The CD4+ T lymphocyte count also were done in the same laboratories.

All the patients were proven cases of HIV seropositivity, and referred from GHTM with various ophthalmic complaints to the Regional Institute of Ophthalmology, Chennai.

Detailed History taking and clinical examination done.

History taking including

- Occupation
- Sexual Habits
- IV Drug abuse
- Socio economic status
- General symptoms and duration
- Ocular symptoms and duration
- Relevant past history
- Sexually transmitted diseases
- History relates to mode of transmission
- Time of HIV detection and presenting illness at that times.

Ophthalmic evaluation includes:

- Visual acuity – Snellen's chart
- Oblique examination with Torch and Loupe
- Detailed slit lamp examination of Anterior Segment
- Slit lamp biomicroscopy with +90 D lens
- Detailed fundus examination – after dilatation with mydriatics – Direct and Indirect Ophthalmoscopy.

OBSERVATION AND RESULTS

Total Number of Patients : 60

Age Distribution

Table No: 1

Age group	No of patients (%)
0-10	-
11-20	1 (1.67%)
21-30	15 (25%)
31-40	35 (58.33%)
41-50	9 (15%)
51-60	-
Total	60

Out of the 60 Patients in this study 50 patients were between 20-40 years of age.

Sex Distribution
Table: 2

Sex	No. of Patients
Male	38 (63.33%)
Female	17 (11.67%)
Eunuchs	5 (8.33%)
Total	60

The Male and Female ratio in this study is 2.24:1

Occupation of HIV+ Patients
Table: 3

Occupation	No. of Patients
Unskilled Labourers	13 (21.67%)
Drivers	22 (36.67%)
Commercial sex workers	7 (11.67%)
others	18 (30%)
Total	60

SEXUAL HABITS OF PATIENTS

Table: 4

SEXUAL HABITS	NO OF PATIENTS
Heterosexual	53 (88.33%)
Homosexual	7 (11.67%)
Total	60

About 90% patients were Heterosexual in this study.

SOURCE DISTRIBUTION

Table: 5

MODE OF TRANSMISSION	NO OF PATIENTS
Trans Sexual	55 (91.67%)
IV Drug abuse	5 (8.33%)
Total	60

Sexual exposure was the commonest mode of transmission in this study.

SYSTEMIC MANIFESTATION

Table: 6

SYSTEMIC MANIFESTATION*	NO OF PATIENTS
RS	42(70%)
CNS	4 (6.67%)
Skin	8 (13.34%)
ENT	39 (65%)
OTHERS	17 (28.34%)

* RS – Respiratory System

CNS- Central Nervous System

ENT – Ear, Nose and Throat

- Some patients were having more than one or two systemic involvement.
- The most common systemic illnesses in these patients were Pulmonary Tuberculosis, followed by Oral Candidiasis and Skin eruptions.

Ocular Symptoms

Table No: 7

Ocular symptoms	No.of patients	Percentage
Defective vision	55	91.67%
Vesicular eruptions	3	5%
Floaters	3	5%
Pain & redness	6	10%
SCH	1	1.67%
Diplopia	4	6.67%
Ptosis	2	3.34%

About 90% patients presented with defective vision ranging from minimal diminision of vision to profound visual loss. About 10% patients presented with pain and redness. Some patients had more than two symptoms.

Time Interval from detection of HIV to Ophthal evaluation.

Table No: 8

Duration (in months)	No. of patients (percentage)
<6	6 (10%)
7-12	17 (28.33%)
13-18	14 (23.33%)
19-24	12 (20%)
25-30	6 (10%)
> 30months	5 (8.33%)

Most of the patients, about 80% were referred within 24 month of detection of HIV infection with ophthalmic symptoms. In patients who were seen in later stages, the symptoms were present even some months earlier and patients seek ophthal opinion later because of poor compliance and other social factors.

Ophthalmic Manifestations

Table No: 9

Orbit and Adnexa		
Herpes zoster Ophthalmicus	3	5%
Kaposi's Sarcoma	1	1.67%
Pthisis bulbi	1	1.67%
Anterior segment		
Anterior uveitis	3	5%
Chronic Panuveitis	6	10%
Chronic Keratouveitis	2	3.34%
Mature Cataract	2	3.34%
Posterior Segment		
CMV Retinitis	16	26.67%
HIV Retinopathy	11	18.33%
Combined RD	2	3.34%
Rhegmatogenous RD	1	1.67%
Progressive Outer Retinal Necrosis	1	1.67%
Acute Retinal Necrosis	1	1.67%
Choroiditis	2	3.34%
Vitritis	2	3.34%
HT Retinopathy	1	1.67%
Neuro ophthal lesions		
Optic Atrophy	5	8.33%
Pappilloedema	5	8.33%
Nerve palsy	4	6.67%

Severity of Visual loss

Table No: 10

Vision in diseased eye	No. of patients (%)
≥ 6/12	9 (15%)
6/18 – 6/60	16 (26.67%)
5/60 – 3/60	4 (6.67%)
2/60 – 1/60	10 (16.67%)
≤ HM	21 (35%)

Out of 60 patients in this study about 50% of patients were having severe visual loss, less than 2/60 is snellen's chart which prevents them being independent socially.

CD4+ T Lymphocyte count in HIV + patients

Table No: 11

CD4+ T cell count	No of patients (%)
< 50 cells / cu.mm	17 (28.33%)
51-100 cells / cu.mm	20 (33.33%)
101-150 cells / cu.mm	12 (20%)
151-200 cells / cu.mm	1 (1.67%)
201 – 250 cells / cu.mm	3 (57%)
> 250 cells / cu.mm	7 (11.67%)

About 60% of patients in this study were having CD4+ T Lymphocyte count less than 100 cells/ cu.mm. Another 20% of patients had CD4+ T Lymphocyte count in the range of 100-150 cells / cu.mm.

Ocular Manifestations, No. of patients and CD4+ T Lymphocyte count.
Table No: 12

Ocular Manifestations	No. of patients	CD4+ Count
Herpes Zoster Ophthalmicus	3	200-250 cells/cu.mm
Kaposi's sarcoma	1	180 cells/cu.mm
Chronic Panuveitis	6	100-150 cells/cu.mm
Chronic Keratouveitis	2	50-100 cells / cu.mm
CMV Retinitis (Total 16 patients)	8 5 1 2	< 50 cells / cu.mm 51-100 cells / cu.mm 101-150 cells / cu.mm > 150 cells / cu.mm
HIV retinopathy (Total 11 patients)	1 9 1	< 50 cells / cu.mm 51-100 cells / cu.mm 101-150 cells / cu.mm
Progressive Outer Retinal Necrosis	1	29 cells / cu.mm
Acute Retinal Necrosis	1	93 cells / cu.mm
Choroiditis	2	< 100 cells.
Nerve palsy (Total 4 patients)	2 2	< 50 cells/cu.mm 100-150 cells / cu.mm
Papilloedema (Total 5 patients)	2 1 2	< 50 cells / cu.mm 100-150 cells / cu.mm 100-150 cells / cu.mm
Optic Atrophy	5	< 50 cells / cu.mm

DISCUSSION

Incidence of ophthalmic manifestations in HIV patients have been reported as 40%-70% in various studies. Almost all HIV patients develop ocular manifestations at some point of their disease period.

This study has been directed towards various ophthalmic manifestations in 60 HIV seropositive patients and correlation with their CD4+T lymphocyte count in a Tertiary Government Hospital in South India.

Majority of the patients in this study were in the age group of 20-40 yrs, which is comparable to the WHO estimate. This is the sexually active age group and the risk of exposure is very high.

The sex incidence in this study was approximately 2:1. Various reports in 2004 state that male to female ratio is nearing equal. But this ratio varies from region to region everywhere. In India as a whole the ratio is 3:1 and in South India the ratio is nearing equal. The incidence in this study can be attributed to various social and cultural factors.

The mode of transmission in most of the cases is trans sexual, around 91.67%. Rest is due to Intra Venous Drug abuse. Most common source among the males was contact with commercial sex workers. Most common source among the women was from their spouses who were HIV positive patients.

The time interval between detection of HIV infection and ophthalmic presentation is an important factor in this study.

Most of the patients visited this hospital with visual complaints, within two years of detection of HIV infection. About 80% of patients developed ophthalmic manifestations within 24 months. Even in the patients, who were turned up late, they developed ocular problems much earlier and they postponed their ophthalmology consultation due to various social factors.

The most common systemic illnesses in these patients were Pulmonary Tuberculosis, followed by Oral Candidiasis and Skin eruptions.

Coming to ocular symptoms, most of the patients have defective vision as their main complaint ranging from mild defective vision to profound visual loss. Most of the patients had floaters and flashes with mild loss of vision in the earlier stages and they ignored these symptoms and landed up as visually handicapped within few months.

Pain and redness were the main complaints in patients with anterior Uveitis, Panuveitis and Chronic Keratouveitis.

Out of 60 patients in this study posterior segment lesions comprised about 50% of ophthalmic manifestations. Most common manifestation is CMV Retinitis in 16 patients (26.67%). Next one is HIV retinopathy in 11 patients (18.33%).

CMV Retinopathy presents as either “Pizza pie “ appearance in most patients and “ Frosted branch angitis” in some patients.

These lesions are evaluated by direct and indirect ophthalmoscopy. According to literature, diagnosis of CMV Retinitis is mainly clinical. Serological investigations are of limited valve and they are available in few laboratories only.

The incidence of posterior segment lesions in this study is comparable with the incidence cited in various studies Internationally.

Eight patients with CMV retinitis had CD4+T Lymphocyte count <50 cells / cu.mm and 5 patients had CD4+ T Lymphocyte count in the range of 50-100 cells / cu.mm which is comparable to WHO reports and various studies. Three patients had CD4+ T Lymphocyte count > 100 cells / cu.mm which may be attributed to the effect of “HAART” therapy, they are undergoing. Most of the patients with CMV retinitis have very poor vision in the range of 6/60 to No Perception of Light.

HIV retinopathy also is evaluated ophthalmocopically. Eleven patients in this study had HIV retinopathy and their CD4+ T Lymphocyte count is less than 100 cells / cu.mm in 10 patients. These patients preserve their vision at the time of presentation, better than 6/36 in most patients.

Two patients had active choriditis lesions. Their antibody levels for Toxoplama were negative. Literature and various studies reveal that antibody titre may be negative due to their immunocompromised state.

Progressive Outer Retinal Necrosis and Acute Retinal Necrosis were seen in one patient each.

Combined Retinal Detachment with both Rhegmatogenous and Tractional components seen in 2 patients most probably due to complication of any posterior segment lesions of HIV diseases.

Chronic panuveitis was noted in 6 patients and anterior uveitis in 3 patients. All these patients have CD4+ T Lymphocyte count within the range of 100-150 cells / cu.mm.

Three patients had Herpes Zoster Ophthalmicus and their CD4+ T Lymphocyte count is in the range of 200-250 cells / cu.mm. One patient with Herpes Zoster Ophthalmicus developed panophthalmitis in one eye and the eye became pthisical within two months of the onset of disease since the patients did not seek proper treatment in time.

One patient had suspected Kaposi’s sarcoma in the form of persistent subconjunctival haemorrhage.

Five patients had papilloedema of both eyes of which 3 patients were having Cryptococcal meningitis proved by lumbar puncture. Two patients had raised intracranial tension with diffuse cerebral cortical atrophy noted in Computed Tomography. All five patients had CD4+ T Lymphocyte count < 150 cells / cu.mm.

Four patients had Nerve palsies in the form of restricted eye movements and Optic atrophy seen in 5 patients, most probably due to complications of posterior segment lesions evidenced by CMV retinitis changes in posterior pole.

SUMMARY

- This clinical study was done at Regional Institute of Ophthalmology, Chennai
- A total of 60 patients who are HIV positive were evaluated, out of that 40 were males and 20 were females.
- Relevant data were collected from each patient and they were subjected to a detailed ophthalmic evaluation.
- 83% belong to the age group of 20-40 years.
- Mode of transmission in most of the patients is Trans sexual, the rest were due to IVD abuse.
- Most of the patients developed ophthalmic complications within 2 years of detection of HIV infection.
- More than 60% of patients were severely affected with the visual activity of less than <6/60.
- More than 50% patients had posterior segment lesions of which the most common manifestation is CMV retinitis followed by HIV retinopathy.
- Most of the patients with posterior segment lesions had CD4+ T Lymphocyte count < 100 cells / cu.mm

- Anterior uveitis and Chronic panuveitis were noted in 15% of patients and CD4+ T Lymphocyte count in these patients is in the range of 100-150 cells / cu.mm .
- 5% patients had Cryptococcal meningitis
- 6.67% patients had Nerve palsies
- 8.33% patients had Optic atrophy.

CONCLUSION

- Ophthalmic manifestations of HIV are increasingly being recognized and the role of an ophthalmologist in its diagnosis.
- Fundus changes are one of the most important ocular manifestations of HIV and can serve as an early indicator for the detection and assessment of progression of disease.
- Routine ophthalmological evaluation should form a part of the clinical examination of all HIV seropositive patients.
- Regular evaluation of CD4+ T Lymphocyte count combined with ophthalmological examination may detect ophthalmic lesions early so that treatment may be started without delay.
- Periodic ophthalmic evaluation in high risk groups like prisoners, commercial sex workers, truck drivers and homosexuals should become mandatory in routine health examination for early detection of AIDS through ophthalmic examination.
- Mass education of the public and increasing their awareness on safe sexual practices, safety precautions during Intravenous injections and Blood transfusions would go a long way in preventing this devastating illness as we all know, PREVENTION IS BETTER THAN CURE.

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KEY TO MASTER CHART

SEX

M – Male

F- Female

use

E – Eunuch

SOURCE

TS – Trans Sexual

IVD – Intra Venous Drug

OCCUPATION

L – Unskilled Labourers

D – Driver

CSW – Commercial Sex

Workers.

O – Others

SEXUAL HABIT

HE – Heterosexual

HO – Homosexual

V/A – Visual Activity

CV – Colour Vision

SYSTEMIC INFECTIONS

PT – Pulmonary Tuberculosis

EPT – Extrapulmonary Tuberculosis

OC – Oral Candidiasis

HZ – Herpes Zoster

HS – Herpes Simplex

GU – Genital Ulcers

GE – Gastro Enteritis

OCULAR SYMPTOMS

DV – Defective Vision

F – Floaters

P – Pain

FIELDS

N – Normal

NP – Not Possible

D – Defective

OPHTHALMIC MANIFESTATIONS.

CWS – Cotton Wool Spots

H – Haemorrhages

MA – Microaneurysm

CKU – Chronic Kerato Uveitis

HZO – Herpes Zoster Ophthalmicus

AU – Anterior Uveitis

CMVR – Cytomegalo Virus Retinitis

CC – Complicated Cataract

CD4 – CD4+ Tlymphocyte Count

N.O. – Neuro Ophthalmology

CPU – Chronic Pan Uveitis

RD – Retinal Detachment

OA – Optic Atrophy

V – Vitritis

MC – Mature Cataract

P – Papilloedema

C- Choroiditis

CASE SHEET

Name: Age / Sex:

IP/OP No: Occupation:

Address:

General symptoms: wt loss/diarrhoea/chronic fever/chronic Cough / oral ulcer/skin lesions

Ocular complaints: defective vision / loss of vision (sudden / Gradual) floaters / flashes / irritation / diplopia /Pain / redness.

Risk factors: sexual promiscuity / previous or present STD infection / IV drug abuse / blood transfusion/CSW

Possible source of infection: transsexual / IV drug / blood transfusion

Time of HIV detection:

Treatment history: since……..at……. as IP / OP

Examination:

General: nourishment / anaemia / jaundice / clubbing

Gen.lymphadenopathy/pedal edema/oral candidiasis.

Pulse: BP:

Systemic examination:

CVS:

RS:

P/A:

CNS:

Associated systemic infections: GI/MC/PT/EPT/HS/HZ/GU/OC/LRI/GE.

Ocular examination:

	RE	LE
Eye brows		
Eye lid		
Lacrimal system		
Conjunctiva		
Sclera		
Cornea		
AC		
Iris		
Pupil		
Lens		

SLE: normal / specific

Ocular kaposis / conjunctival candidiasis

Ocular herpes / ocular molluscum

V/A:

Colour vision

Fields

Fundus

Direct

IDO:

(CMV retinitis / optic neuritis crptococcal / pneumocystic carinii chorioretinitis)

TC: DC: ESR: Hb:

RBS: blood urea: VDRL:

Serology for HIV / ELISA / TRIDOT / WB

CD4 Count:

Sputum for AFB:

Others

Follow up:

KEY TO MASTER CHART

SEX

M – Male

F- Female

use

E – Eunuch

SOURCE

TS – Trans Sexual

IVD – Intra Venous Drug

OCCUPATION

L – Unskilled Labourers

D – Driver

CSW – Commercial Sex

Workers.

O – Others

SEXUAL HABIT

HE – Heterosexual

HO – Homosexual

V/A – Visual Activity

CV – Colour Vision

SYSTEMIC INFECTIONS

PT – Pulmonary Tuberculosis

EPT – Extrapulmonary Tuberculosis

OC – Oral Candidiasis

HZ – Herpes Zoster

HS – Herpes Simplex

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OCULAR SYMPTOMS

DV – Defective Vision

F – Floaters

P – Pain

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NP – Not Possible

D – Defective

OPHTHALMIC MANIFESTATIONS.

CWS – Cotton Wool Spots

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CPU – Chronic Pan Uveitis

RD – Retinal Detachment

OA – Optic Atrophy

V – Vitritis

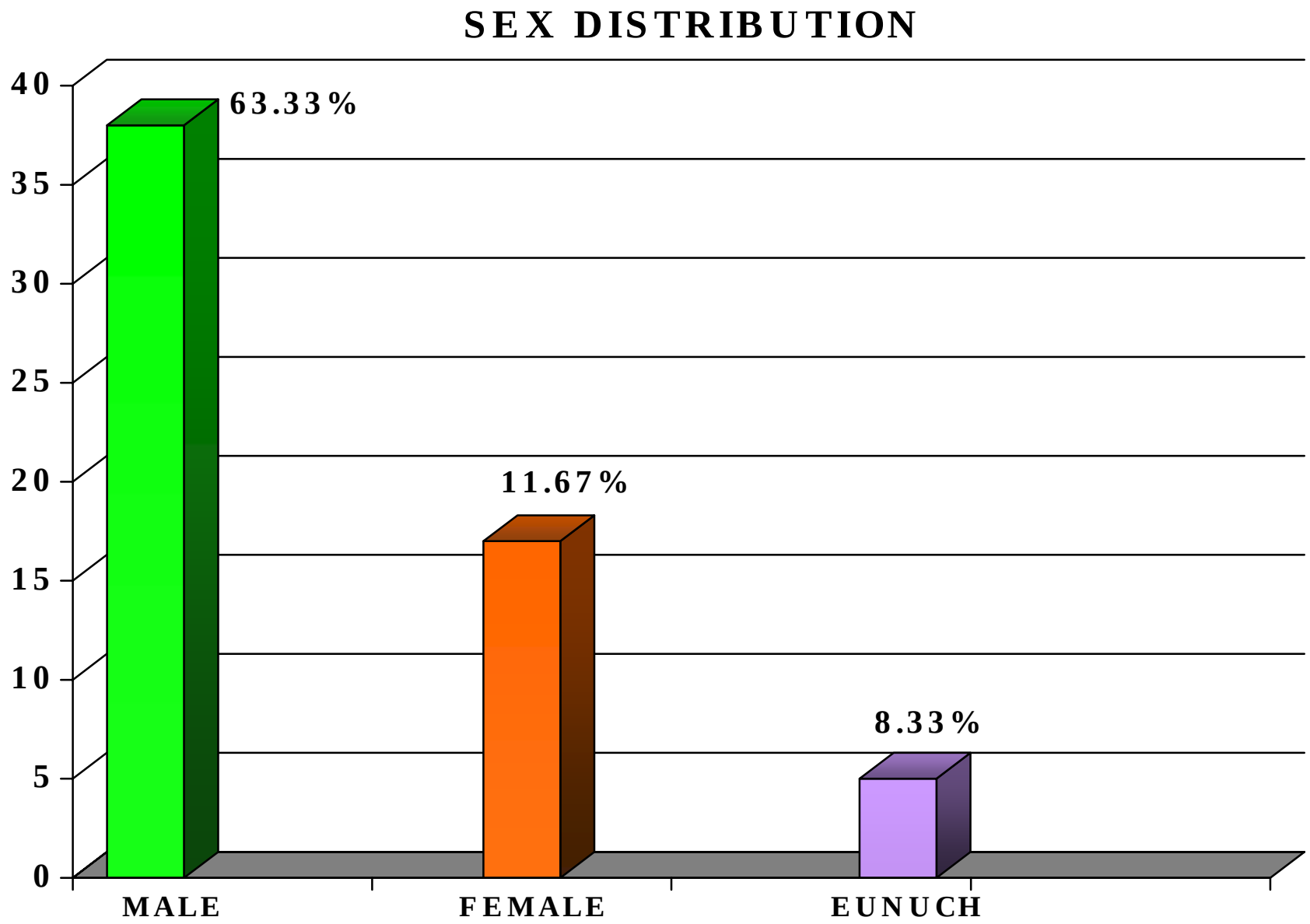
MC – Mature Cataract

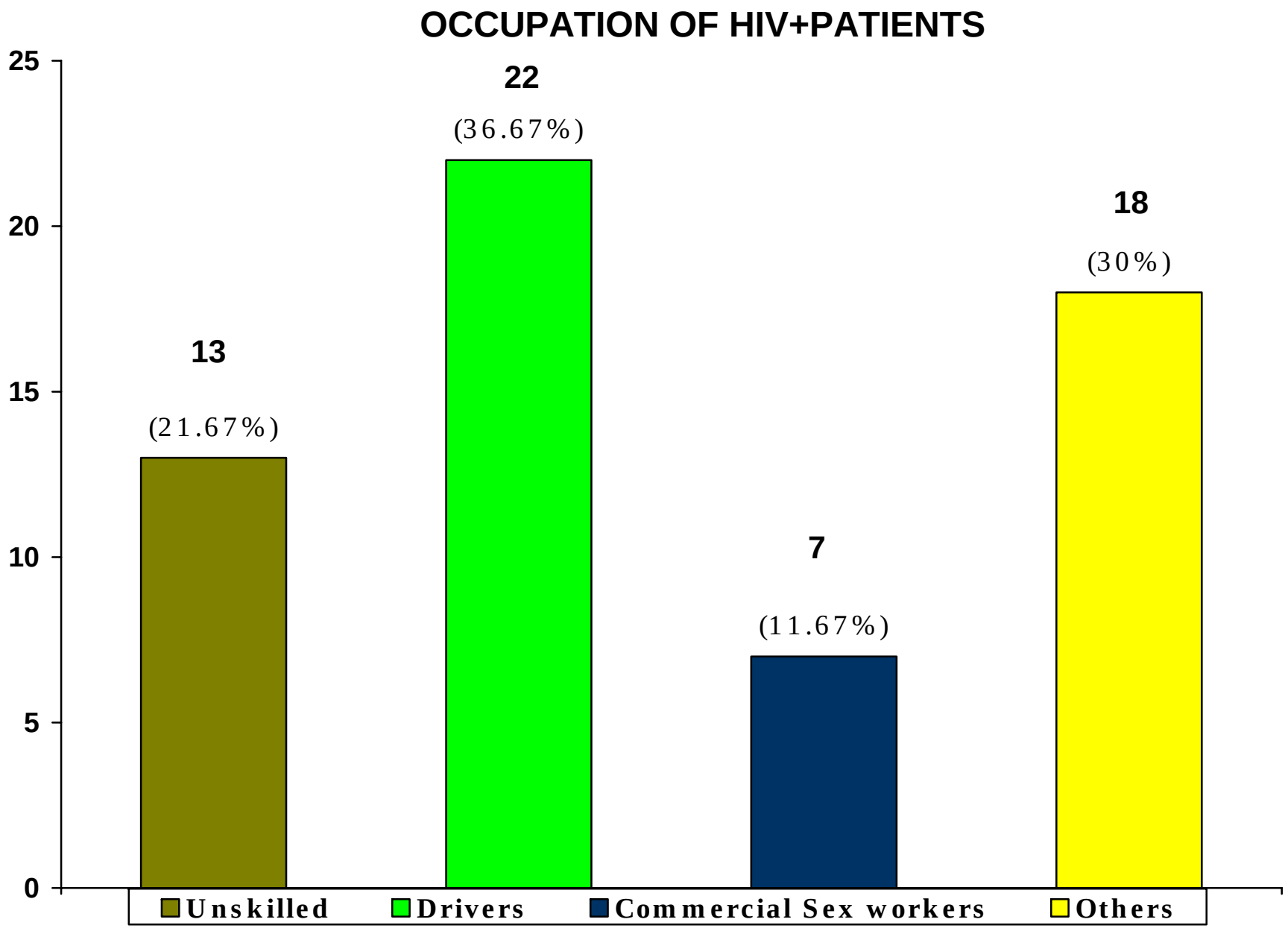
P – Papilloedema

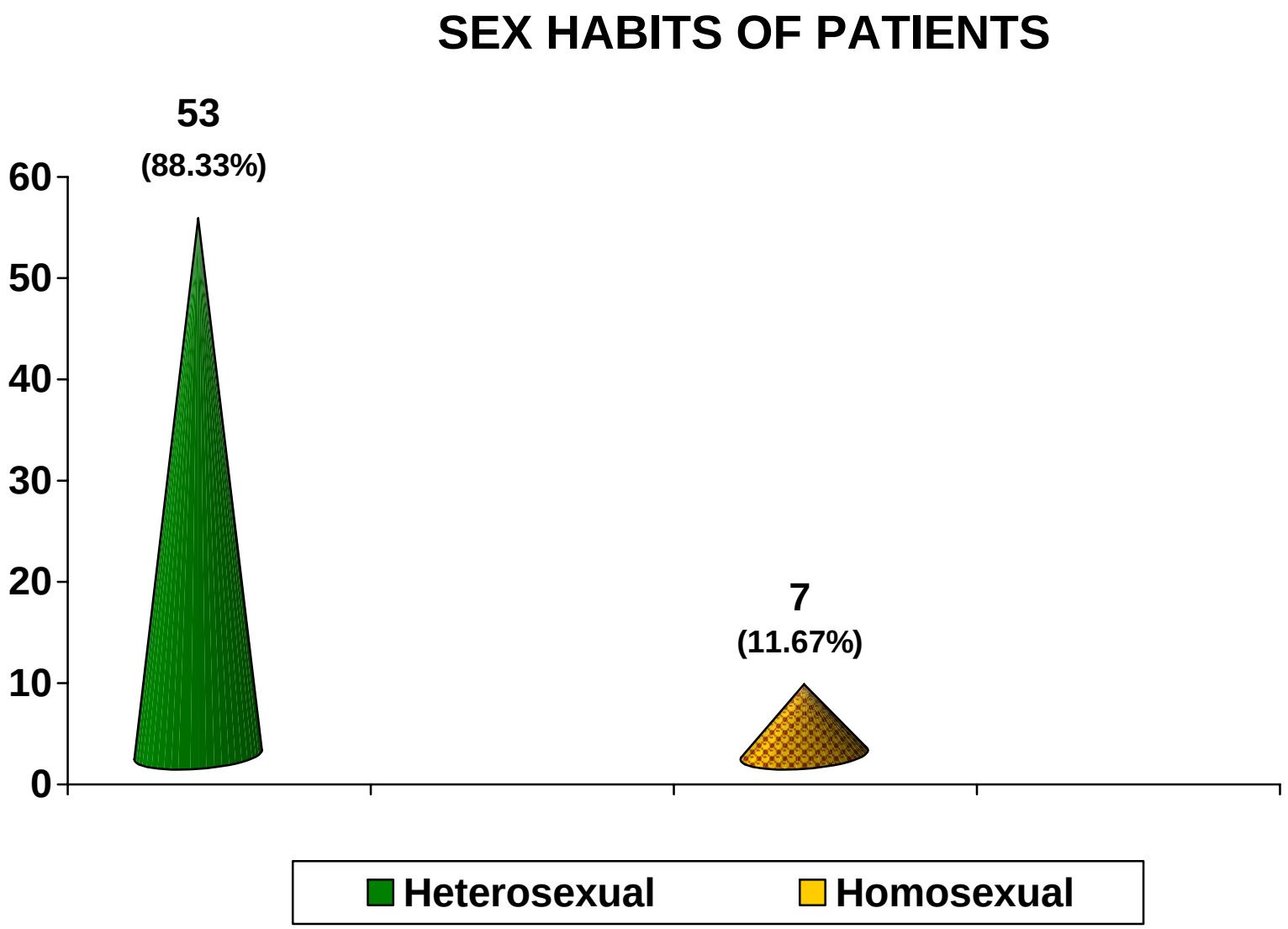
C- Choroiditis

LIST OF OPERATIONS PERFORMED

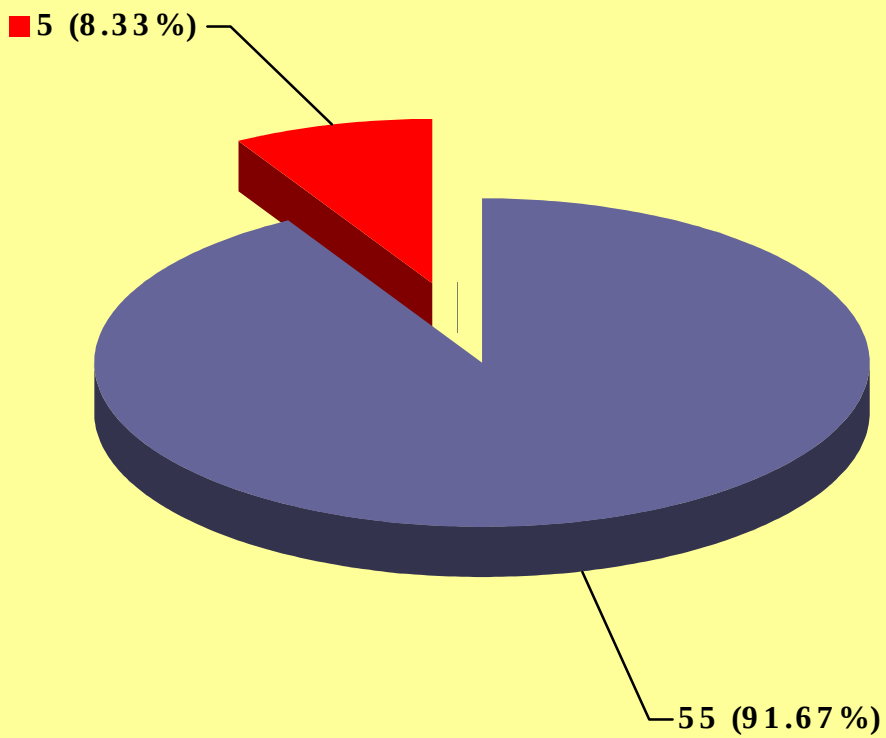
SI.No	Hospital No	Date	Name	Age/Sex	Surgery
1	379005	27/03/04	Vijayalakshmi	45/F	RE ECCE with PI
2	381853	18/06/04	Angamuthu	65/F	LE ECCE with PCIOL
3	386915	04/12/04	Marimuthu	45/F	RE ECCE with PCIOL
4	391184	25/04/05	Janakiammal	52/F	RE SICS with PCIOL
5	392342	30/05/05	Kamatchi	60/F	RE SICS with PCIOL
6	701231	17/11/05	Mangammal	67/F	RE ECCE with PCIOL with Trabeculectomy
7	711201	15/12/05	Jaggamma	72/F	RE ECCE with PCIOL with Trabeculectomy
8	722307	12/01/06	Lourdus	68/M	RE RD Surgery
9	385815	17/10/04	Subbarayan	65/M	RE Therapeutic Keratoplasty
10	389714	17/11/04	Senthamarai	45/F	LE Therapeutic Keratoplasty
11	29713	05/05/04	Kavitha	17/F	RE Chalazion Incision and Drinage
12	48656	30/06/04	Muniamma	68/F	LE Ptergium Excision
13	67514	01/02/05	Arumugam	65/M	LE Dacryocystectomy
14	7940	04/02/06	Poongothai	25/F	RE Dacryo cysto rhinostomy
15	27154	27/11/04	Govindhan	43/M	LE Lower lid tear suturing
16	1751	06/02/05	Angamma	63/F	RE Evisceration
17	2315	28/03/05	Pappammal	67/F	RE Tarsorrhaphy
18	4375	20/05/05	Somu	47/M	LE Corneal tear suturing
19	1327	18/02/06	Madhavan	53/M	RE Corneo scleral tear suturing
20	41477	20/05/05	Kesavaiah	67/M	RE Scleral tear suturing


SEX DISTRIBUTION
40
35
30
25
20
15
10
5
0
63.33%
11.67%
8.33%
MALE
FEMALE
EUNUCH

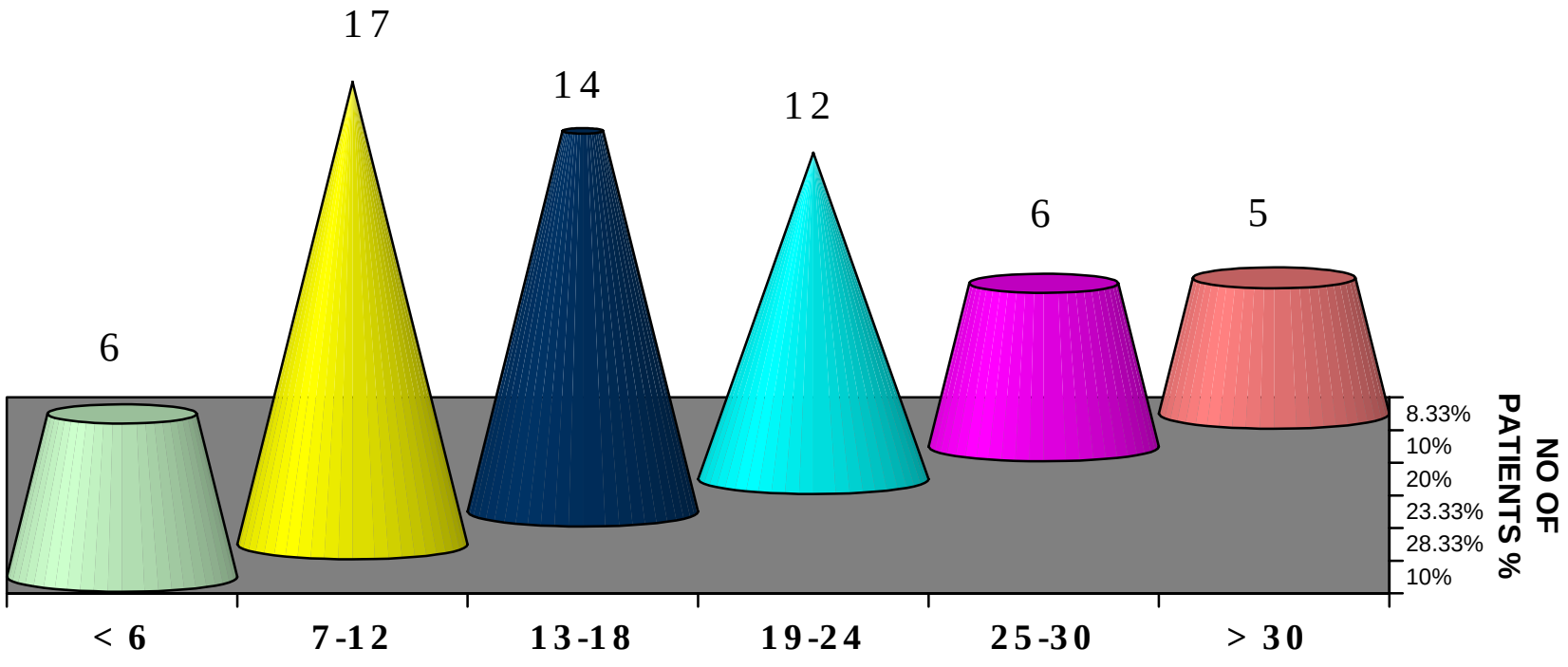

OCCUPATION OF HIV+PATIENTS
25
20
15
10
5
0
22
(36.67%)
18
(30%)
13
(21.67%)
7
(11.67%)
Unskilled
Drivers
Commercial Sex workers
Others


SEX HABITS OF PATIENTS
53
(88.33%)
7
(11.67%)
60
50
40
30
20
10
0
Heterosexual
Homosexual

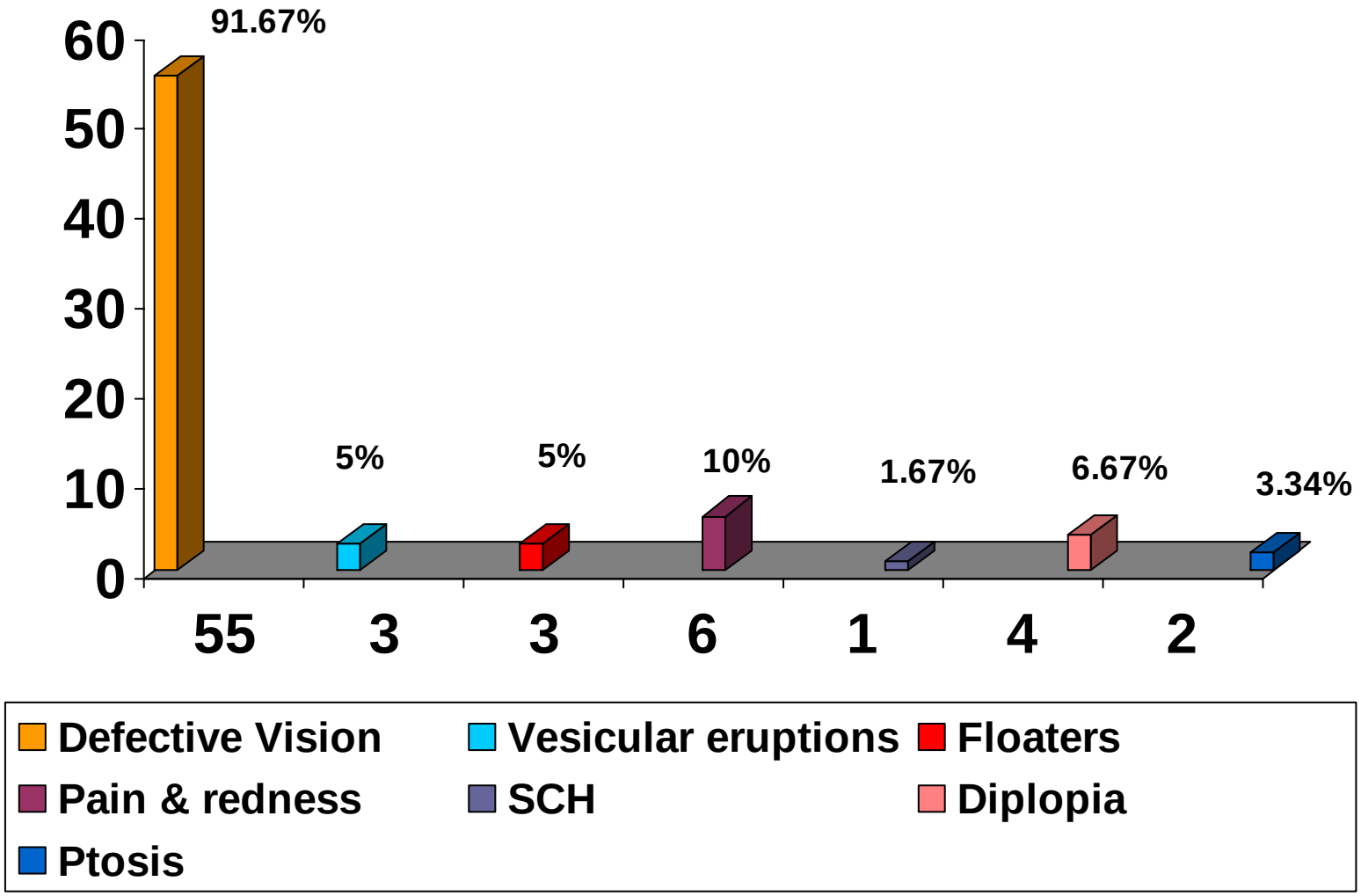
SOURCE DISTRIBUTION

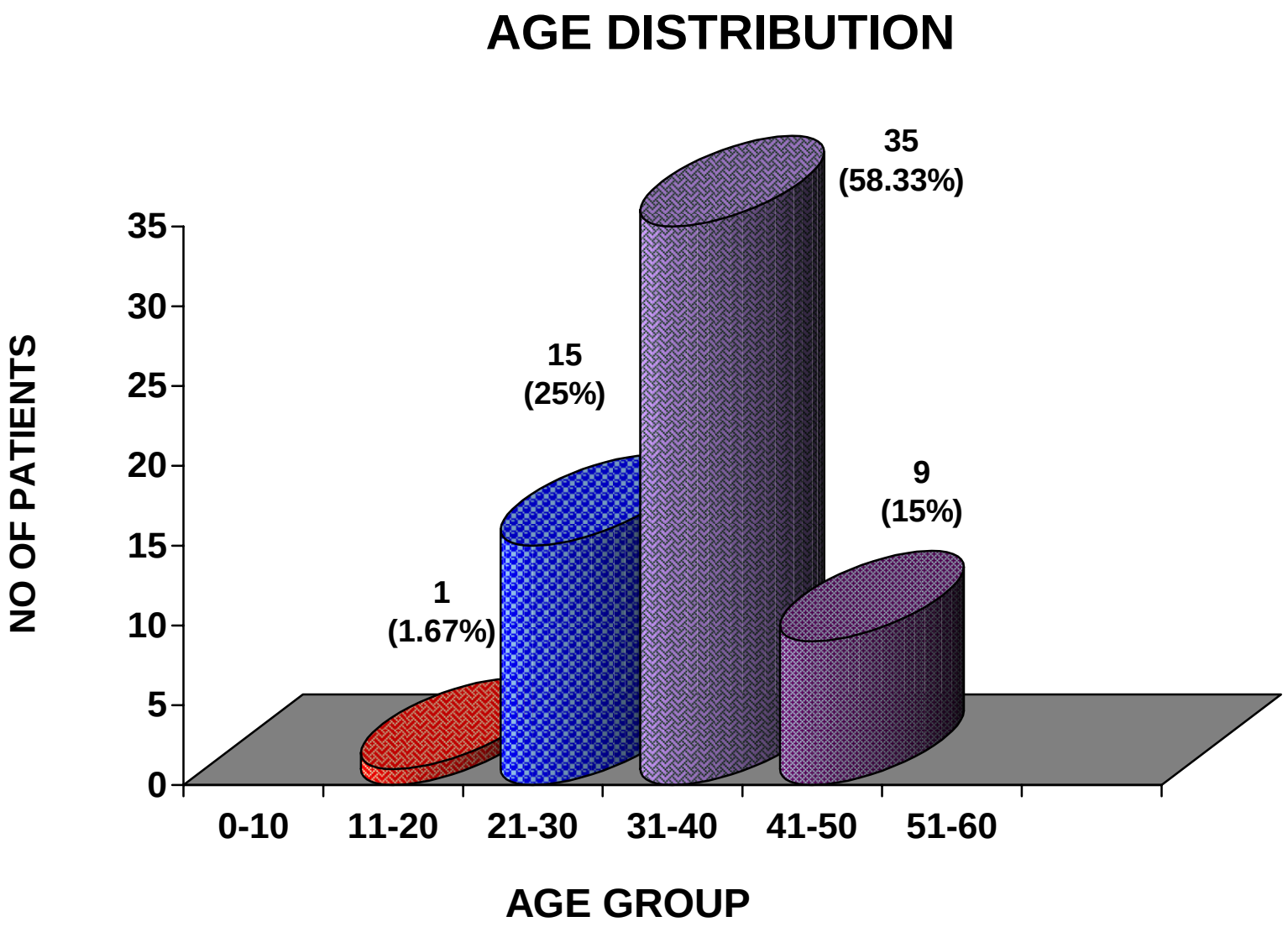


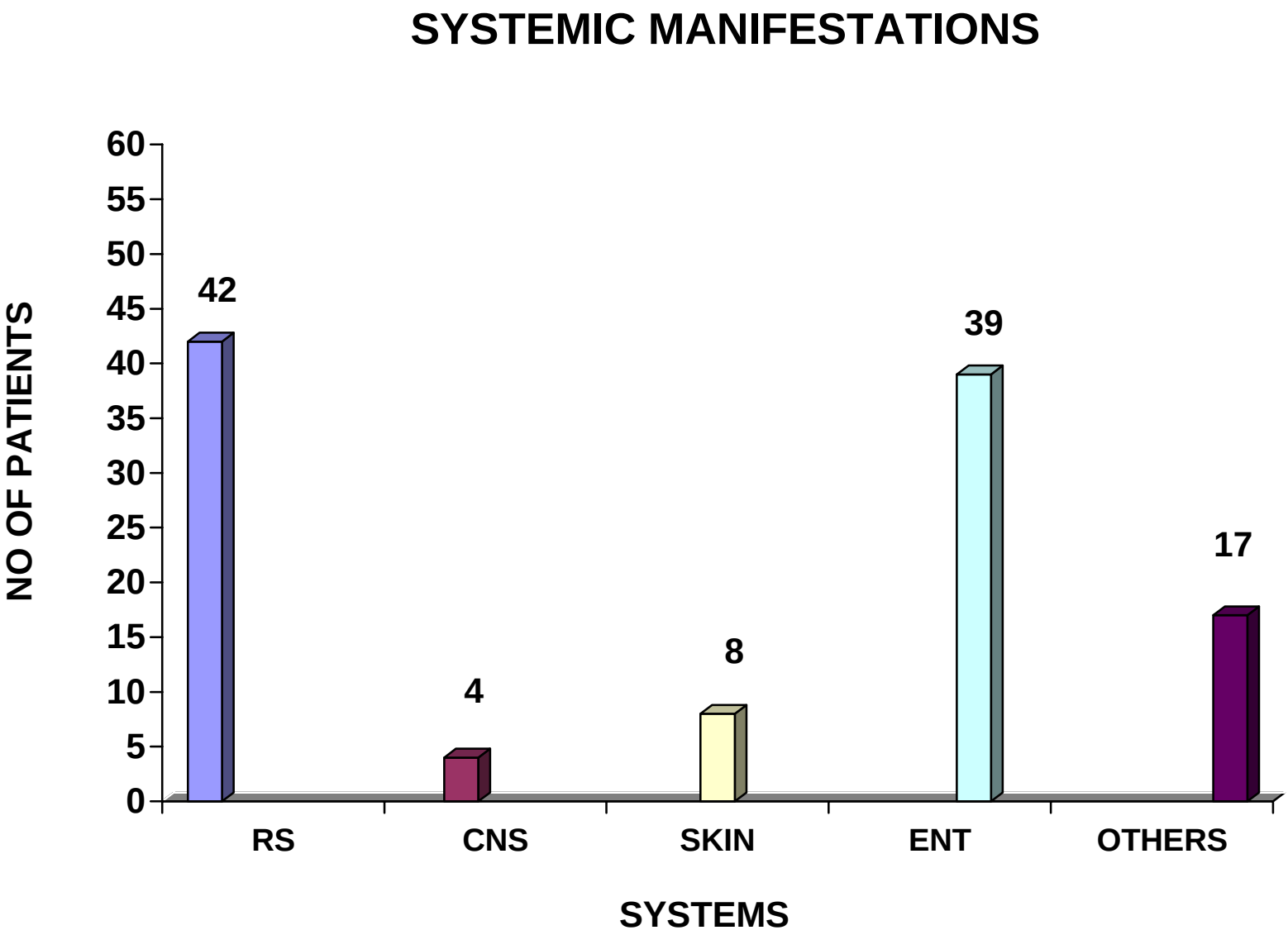
TIME INTERVAL FROM DETECTION OF HIV TO OPHTHAL AFFECTATIONS



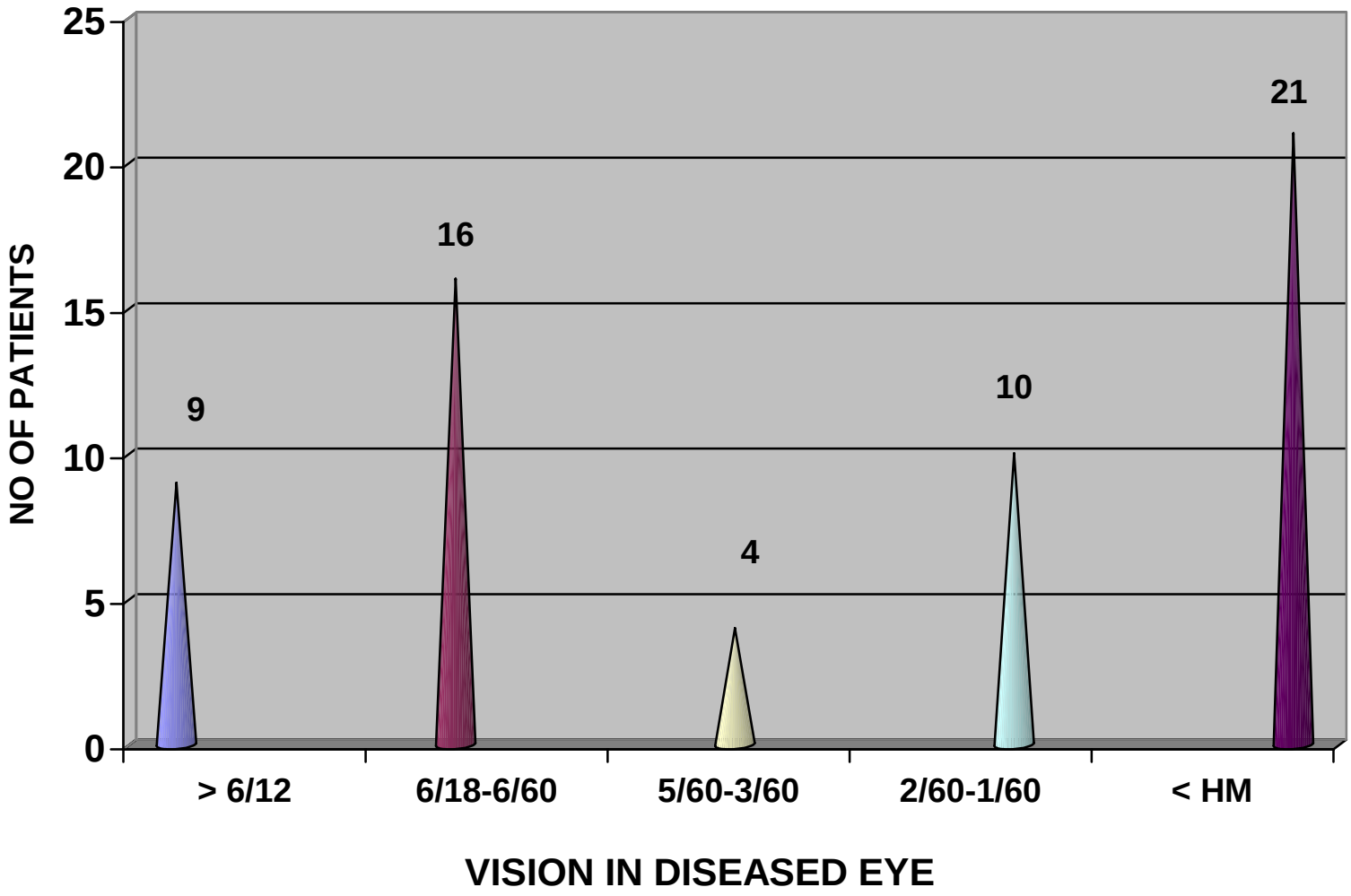
OCULAR SYMPTOMS

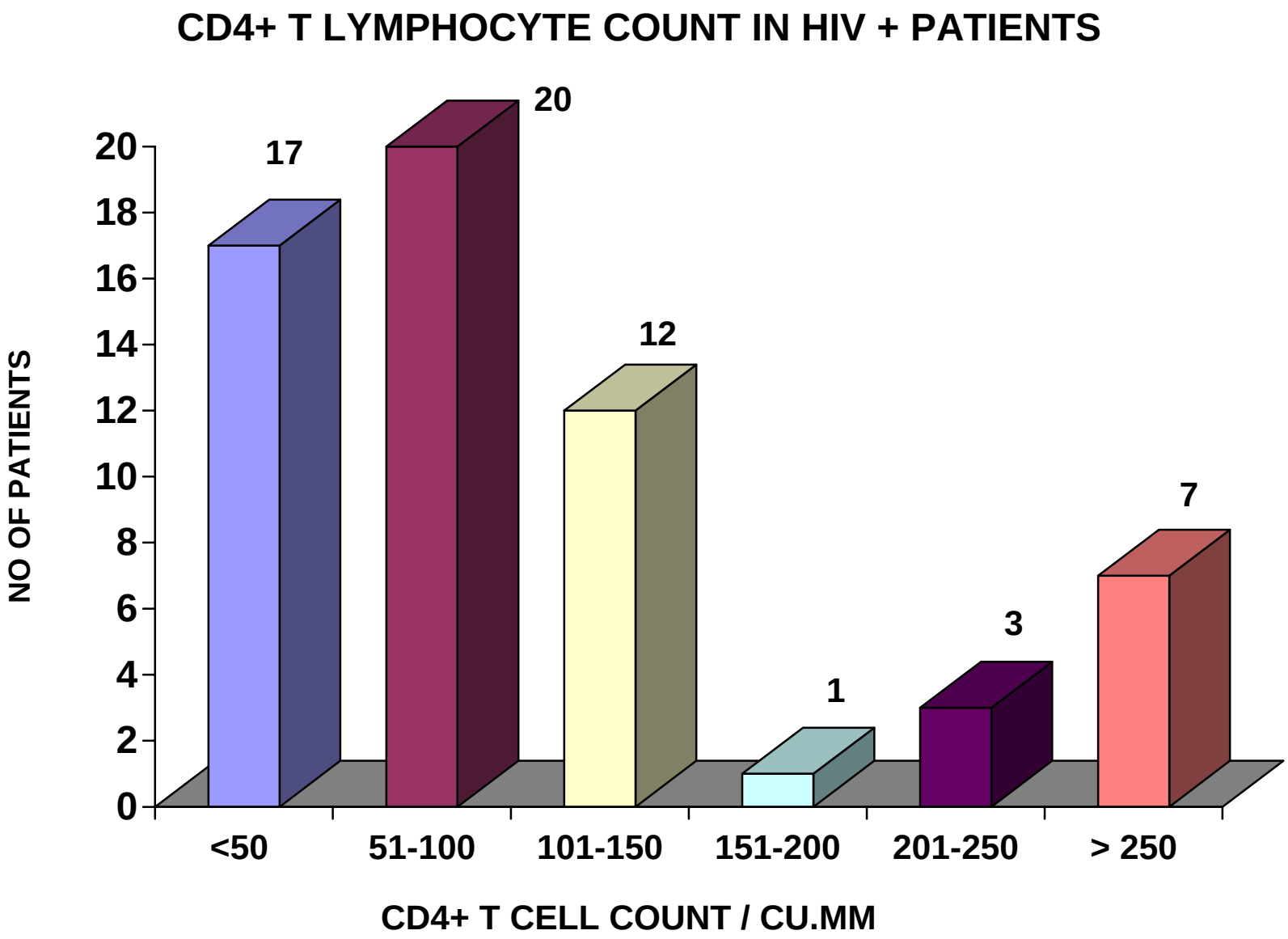



AGE DISTRIBUTION
NO OF PATIENTS
35
30
25
20
15
10
5
0
1
(1.67%)
15
(25%)
35
(58.33%)
9
(15%)
0-10
11-20
21-30
31-40
41-50
51-60
AGE GROUP

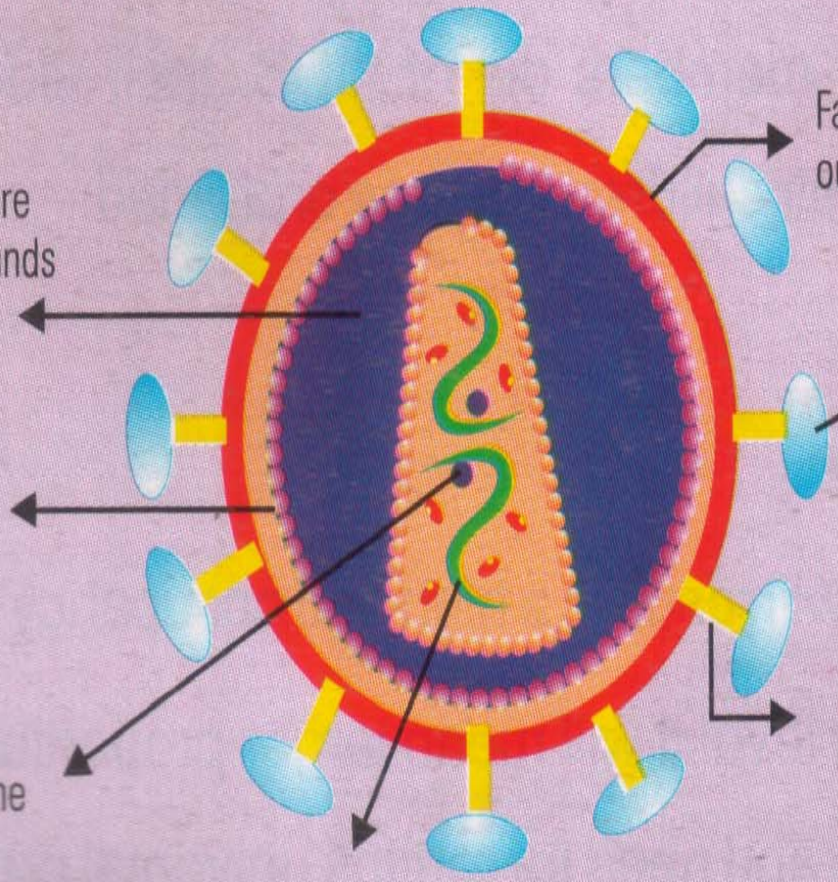

SYSTEMIC MANIFESTATIONS
NO OF PATIENTS
60
55
50
45
40
35
30
25
20
15
10
5
0
42
4
8
39
17
RS
CNS
SKIN
ENT
OTHERS
SYSTEMS

SEVERITY OF VISUAL LOSS




CD4+ T LYMPHOCYTE COUNT IN HIV + PATIENTS
NO OF PATIENTS
0
2
4
6
8
10
12
14
16
18
20
17
20
12
1
3
7
<50
51-100
101-150
151-200
201-250
> 250
CD4+ T CELL COUNT / CU.MM

Structure of Human Immunodeficiency Virus



Fat layer - the outer coat of the virus

P-24 - a protein in the core of the virus which surrounds the RNA

GP-120 - sugar and protein combination that helps the virus get attached to cells and attack them

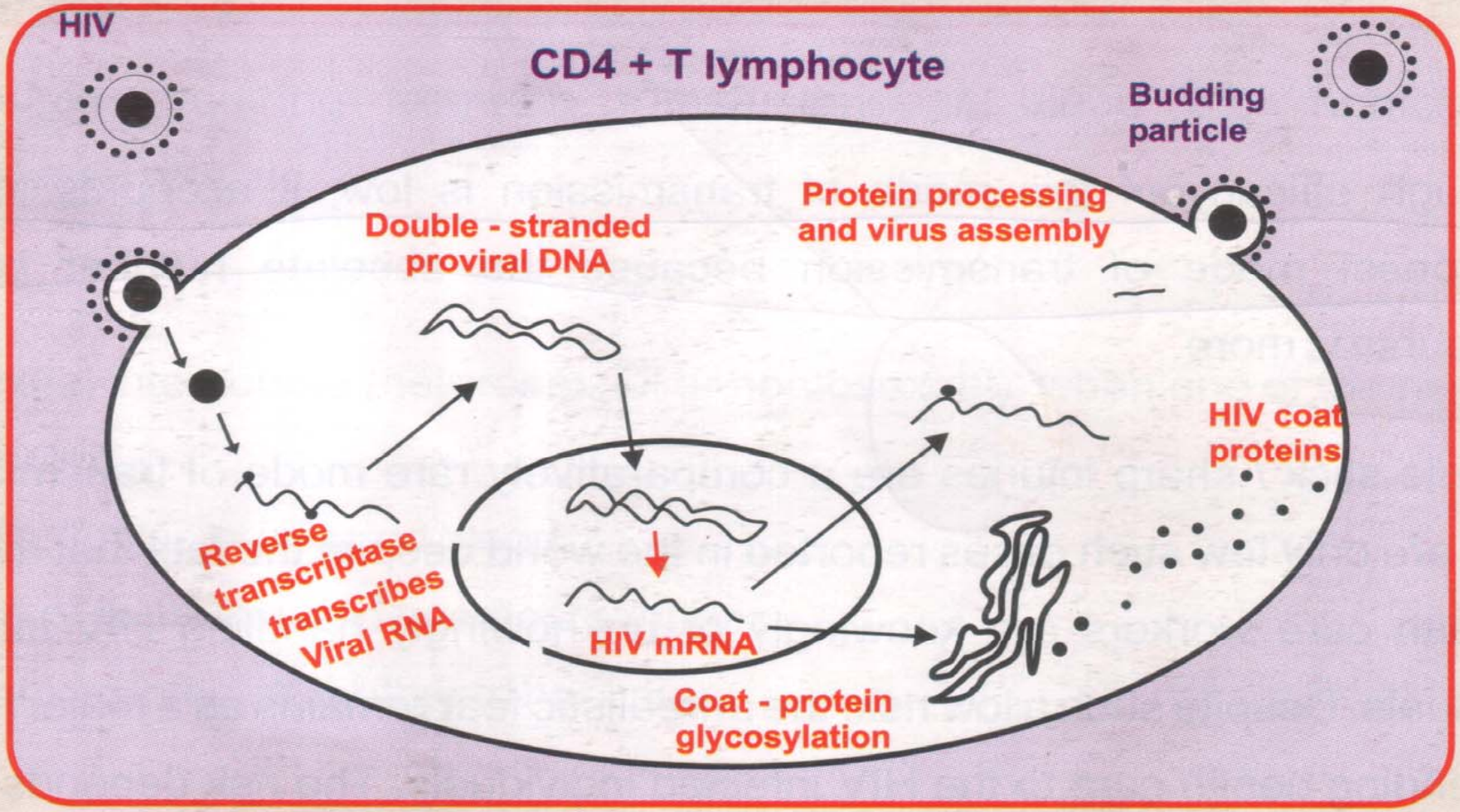
P-17 - a protein in the inner part of the virus that is believed to produce viral surface

GP-41 - sugar and protein combination that helps the outer coat to attack the cells.

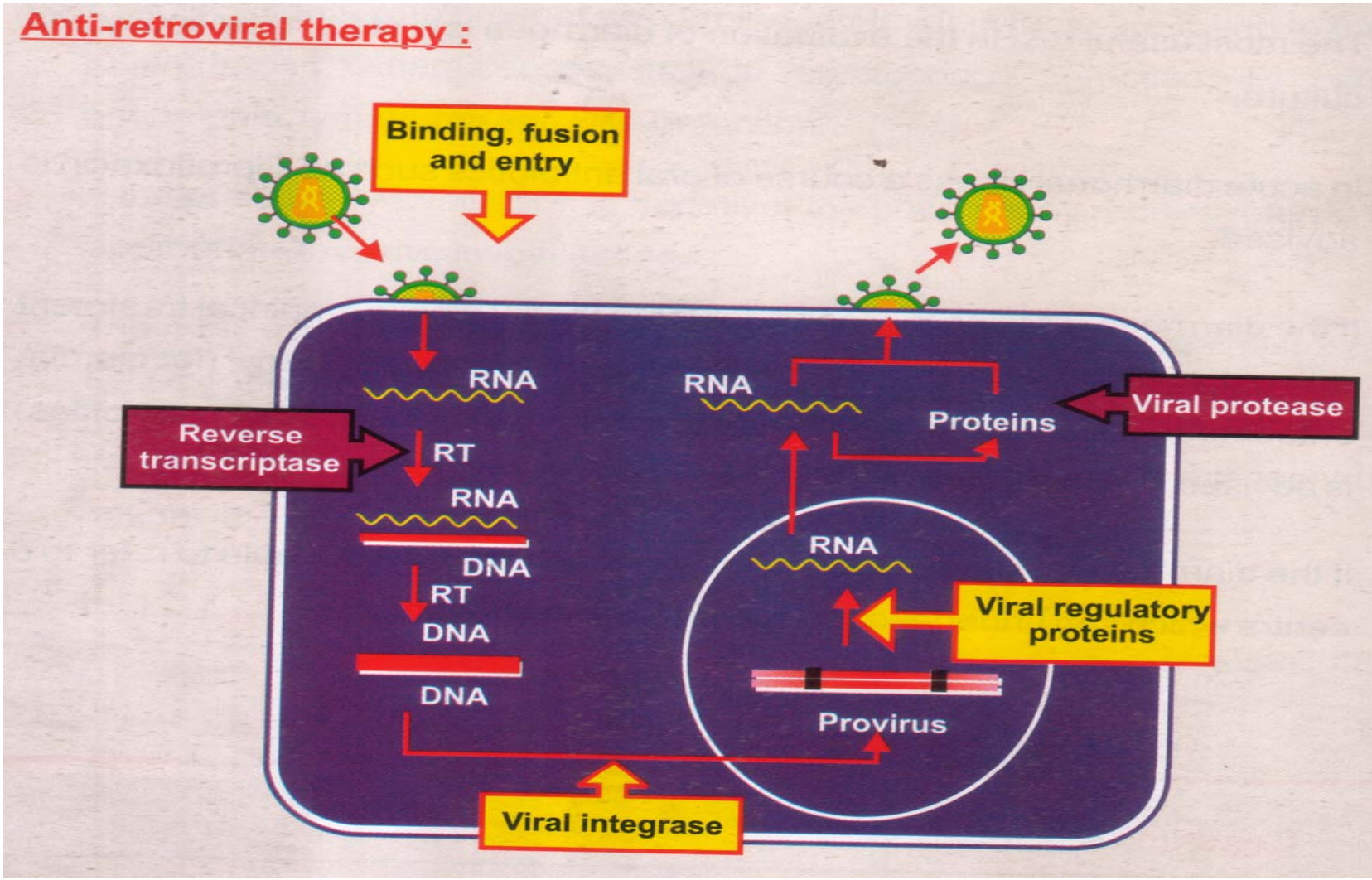
Reverse transcriptase - the enzyme that helps the virus take over cells

RNA - the genetic material

HIV REPLICATION IN THE CELL



ENZYMES INHIBITING SITES



S.No.	Name	Age	Sex	Occupation	Sexual Habits	Source	Duration in Months	Systemic Illness	V/A	Complaint	CD4 Count	Orbit / Adnexa	Anterior Segment	Posterior Segment	N.O	Fields
1	Pazhani	37	M	L	HE	TS	10 Mths	GI,PT,OC,GE	6/36,6/60	DV	349			Gr.IV HT.RP		N
2	Govindhan	36	M	D	HE	TS	22 Mths	PT,OC	6/18,6/18	F,DV	286		AU			N
3	Padmavathy	38	F	O	HE	TS	7 Mths	OC	2/60,4/60	DV,PAIN	123		CPU,CC	Tr.with Rheg.RD		NP
4	Vasantha	36	F	CSW	HE	TS	16 Mths	PT,OC	PL+,6/18	DV	24			CMVR, OA		NP
5	Sridhar	34	M	D	HE	TS	26 Mths	OC,GE	6/12,6/9	DV	412			V		N
6	Ramu	40	F	O	HE	TS	28 Mths	PT,HS,GU	1/60,PLP	DV	252		MC,MC			NP
7	Revathy	26	F	L	HE	TS	9 Mths	PT,OC, GU	6/12,6/18	DV	107			P(BE), VI NP		N
8	Jegan	35	M	D	HO	TS	18 Mths	GE	HM,6/6	DV	87			CMVR		NP
9	Marimuthu	40	M	O	HE	TS	20 Mths	OC	2/60,3/60	DV	16			CC		NP
10	Gandhimathi	35	F	O	HE	TS	11 Mths	PT	6/12,6/18	DV	24		AC			N
11	Paliah	16	M	D	HE	TS	6 Mths	PT,OC	6/18,6/24	DV	76			CMVR		N
12	Anbazhagan	40	E	CSW	HO	TS	38 Mths	PT,OC	6/12,6/12	DV	87			CWS/MA		N
13	Mallika	37	F	L	HE	TS	8 Mths	EPT	6/60,2/60	DV	74			CMVR		NP
14	Madhavan	34	M	D	HE	TS	36 Mths	OC	NOPL,NOPL	DV	10			OA, III NP		NP
15	Azhagesan	32	M	O	HE	TS	5 Mths	PT	6/6,NOPL	DV	211	PTHYSIS				NP
16	Anbu	24	M	D	HE	TS	9 Mths	PT,OC	6/12,1/60	DV	126		CPU			NP
17	Balan	43	E	CSW	HO	TS	8 Mths	PT,OC	PL+, PL+	PAIN	113		CPU,CC			NP
18	Muthu	28	M	L	HE	IVD	2 Mths	PT	HM,6/19	DV PAIN	89		CKU			NP
19	Gangadharan	35	M	D	HE	TS	13 Mths	OC,PT	PL+, PL+	DV	36				P(BE)	NP
20	Ramaththa	30	F	O	HE	TS	16 Mths	PT	6/24,6/24	DV	76			CWS/MA		N
21	Mohan	30	M	D	HE	TS	24 Mths	OC	6/6, CFCF	DV	252			RD		NP
22	Thirupathy	45	M	D	HE	TS	48 Mths	PT	6/12,6/12	DV	30			CWS/MA		N
23	Malathi	42	F	O	HE	TS	6 Mths	HZ,OC	HM, 6/12	DV	45			CMVR	OA	NP
24	Ekeswaran	40	M	D	HO	TS	7 Mths	EPT,OC	6/18,6/12	DV	9			CMVR		N
25	Subani	32	M	D	HE	TS	11 Mths	OC,PT	NO PL, HM	DV	27			CMVR	OA	NP
26	Selvi	31	F	L	HE	TS	14 Mths	PT	6/60,6/60	VESICLES	29			PDRN		N
27	Nagarajan	38	M	D	HE	TS	15 Mths	OC	6/18,6/12	DV	267	HZO				N
28	Yasodha	24	F	O	HE	TS	18 Mths	PT,OC	6/12,6/24	DV	87			CWS/MA		N
29	Krishnan	43	M	O	HE	TS	22 Mths	HZ,OC	6/12,6/18	DV	67			CWS/MA		N
30	Kumar	35	M	D	HE	TS	9 Mths	OC	2/60,3/60	DV	112			CMVR		NP

S.No.	Name	Age	Sex	Occupation	Sexual Habits	Source	Duration in Months	Systemic Illness	V/A	Complaint	CD4 Count	Orbit Adnexa	Anterior Segment	Posterior Segment	N.O	Fields
31	Rajan	37	M	L	HE	IVD	13 Mths	PT,OC	3/60,1/60	DV	93			ARN		NP
32	Selvam	32	M	O	HE	TS	16 Mths	PT,OC	6/12,6/24	DV	83			CWS		N
33	Leela	26	E	CSW	HE	TS	27 Mths	OC	6/9,6/12	DV	92			CWS		N
34	Jebamani	28	M	L	HE	TS	18 Mths	PT	6/9,6/12	VESICLES	212	HZO				N
35	Valavan	41	M	O	HE	TS	17 Mths	PT,OC	2/60,3/60	DV	40			CMVR		NP
36	Kondaiah	35	M	D	HE	TS	18 Mths	PT	6/9,6/12	DV,F	73			CWS		N
37	Pugalendhi	37	M	L	HE	TS	10 Mths	PT	PL+,PL+	DV	26			CMVR	OA	NP
38	Mahendran	27	M	D	HE	IVD	22 Mths	EPT	2/60,4/60	DV	64			CMVR		N
39	Dhas	24	M	L	HO	TS	26 Mths	HZ, PT	6/12,6/18	DV	107			CWS		N
40	Natarajan	33	M	D	HE	TS	20 Mths	PT, OC	6/6,1/60	DV	20			CMVR		NP
41	Ismail	38	M	O	HE	TS	11 Mths	PT,OC	6/24,6/24	DV	97			CWS/MA		N
42	Peter	33	M	O	HE	TS	6 Mths	PT, HS	PL+ PL+	DV	48				P(BE)	N
43	Jayarani	28	F	CSW	HE	TS	34 Mths	OC	HM 6/9	DV,PAIN	86		CKU			NP
44	Vellaiammal	47	F	O	HE	TS	9 Mths	PT	PL+ PL+	DV	123		CPU,CC			N
45	Irudayam	31	M	D	HE	TS	8 Mths	EPT	6/12,1/60	DV	107		CPU,CC			
46	Pakayaraj	32	M	L	HE	TS	13 Mths	PT, OC	6/9,6/12	SCH	180	KS				NP
47	Ayyavoo	37	M	D	HO	TS	45 Mths	OC, PT	NOPL,NOPL	DV	20			OA,III NP		N
48	Periaswamy	46	M	L	HE	TS	16 Mths	PT	6/60,6/60	DV	87			CMVR		N
49	Sadayan	35	M	D	HE	TS	15 Mths	OC	6/18,6/12	DV	74			CWS/MA		N
50	Vijaya	26	E	CSW	HE	TS	22 Mths	PT, OC	6/9,6/9	PAIN	112		AU			NE
51	Saroja	31	F	O	HE	TS	27 Mths	PT, OC	3/60,2/60	DV	82			CMVR		
52	Rangarajan	22	M	O	HE	IVD	12 Mths	PT, OC	1/60,2/60	DV	92			C C		NP
53	Chandran	34	M	D	HE	TS	6 Mths	OC. PT	HM,6/6	DV	73			CMVR		NP
54	Rukmani	42	F	O	HE	TS	10 Mths	PT,OC	6/12,6/9	DV	45			V		NP
55	Arumugam	27	M	D	HE	IVD	28 Mths	PT	1/60,2/60	DV	212		MC MC			NP
56	Kuppabai	35	F	L	HE	TS	19 Mths	PT	6/12,6/18	DV	114			P(BE) VI NP		NP
57	Akilandam	37	F	O	HE	TS	22 Mths	OC	PL+ 6/18	DV	24			CMVR	.	N
58	Indrani	29	F	CSW	HE	TS	20 Mths	PT,OC	2/60,4/60	PAIN DV	137		CPU			NP
59	Kannappan	33	M	D	HO	TS	24 Mths	PT, OC	1/60,6/60	DV	274			TR.RhegRD		N
60	Filomina	38	F	L	HE	TS	12 Mths	OC,PT	6/36,6/60	DV	327			CMVR		NP